### POSTTRAUMATIC STRESS DISORDER

### A COMPREHENSIVE TEXT

#### **EDITED BY**

#### PHILIP A. SAIGH

Graduate School and University Center of the City University of New York

#### J. DOUGLAS BREMNER

Yale University School of Medicine and Veterans Administration Connecticut Health Care System West Haven Campus

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# PSYCHOPHYSIOLOGICAL ASSESSMENT OF POSTTRAUMATIC STRESS DISORDER\*

EDWARD B. BLANCHARD, PH.D., AND TODD C. BUCKLEY, B.S. Center for Stress and Anxiety Disorders
University at Albany-SUNY

With the reintroduction of posttraumatic stress disorder (PTSD) into psychiatric nosology by DSM-III (American Psychiatric Association, 1980), steadily growing attention has been paid to the possible role of psychophysiological measures in the assessment of the disorder. This attention to psychophysiology was based in part on the formal definitions of PTSD. Thus, DSM-III called attention (p. 236) to "symptoms of excessive autonomic arousal" but did not specifically include increased physiological responding in the formal diagnostic criteria. DSM-III-R (American Psychiatric Association, 1987) formally included increased physiological responding as one of 17 symptoms which define PTSD: "physiologic reactivity upon exposure to events that symbolize or resemble an aspect of the traumatic event" (p. 250). More recently, DSM-IV (American Psychiatric Association, 1994) retained the symptom, "physiological reactivity on exposure to internal or external cues that symbolize or resemble an aspect of the traumatic event" (p. 428), but classified it to a reexperiencing symptom instead of a hyperarousal symptom in DSM-III-R.

#### **DOBBS AND WILSON (1960)**

Empirical study of this phenomenon or symptom began prior to the publication of DSM-III (or even DSM-II [American Psychiatric Association, 1968])

with the pioneering study of Dobbs and Wilson (1960). Their study, which served as a prototype for much of the subsequent research, involved two groups of World War II (n = 19) and Korean War (n = 2) veterans: a group of 8 male combat veterans who were described as "decompensated" and suffering from "combat neurosis" and a group of 13 male veterans of approximately the same age who had had combat experience but were described as "compensated." A comparison group of 10 university students who had no combat experience and who were all younger than any of the veterans was also included. Cardiac rate (heart rate: HR), respiration rate (RR), and EEG were recorded continuously during the assessment. Subjects first lay quietly for 5 to 7 minutes during a resting baseline; this was followed by 8 minutes of exposure to combat related sounds (artillery bombardment, small arms fire, and aerial bombardment). Finally, flashing lights (to depict explosions) were added to the last 4 minutes.

Interestingly, the "decompensated" veterans (who probably had severe PTSD) were so aroused and upset by the auditory and visual stimuli that no psychophysiological data were recorded from them. Comparisons of the "compensated" veterans to the university students showed the former to have higher baseline HR and RR than the students and also to show greater within session change to the provocative stimulus (6 beats per minute [bpm] versus 2 bpm, respectively, in HR; see Table 12.1 below). One might suspect from the psychophysiological data that the

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"compensated" veterans had, at the very least, some lingering PTSD symptoms.

#### THE DATA BASE

In summarizing the literature on the use of psychophysiological assessment with PTSD, we were able to locate 31 reports. We have included both published reports and papers presented at meetings for which copies of the paper or a detailed abstract were available. We have not included numerous reports on other aspects of the psychobiology of PTSD. Instead, we have limited our coverage to reports in which non-invasive measures, that is, physiologic responses measured with surface sensors, were taken on patients with PTSD under at least two conditions: at least one condition reminiscent of the trauma and one other comparison (non-trauma-related) condition (e.g., resting baseline, non-trauma-related stimulus, etc.). (We have also included three studies of baseline measures only on participants with PTSD and a comparison group without PTSD.) Many studies like the three mentioned above have also included comparison groups of subjects (e.g., normal controls, individuals exposed to the trauma who did not develop PTSD, individuals with other psychiatric disorders, etc.).

Important methodological details and experimental findings from these reports are summarized in Tables 12.1 and 12.2. In Table 12.1 are summarized the reports for which the traumatic event was exposure to combat. They are arranged chronologically in order of date of publication. In Table 12.2 are summarized the reports in which the participants had been exposed to traumatic events other than combat. These latter reports are organized around type of trauma, motor vehicle accidents, rape, or mixed civilian trauma and then presented chronologically.

Most of the remainder of this chapter is a discussion and conclusions one can draw from the data in Tables 12.1 and 12.2, organized around rhetorical questions.

#### WHO HAS BEEN STUDIED?

Examining the two tables, several dominant research themes emerge. Two-thirds (21/31) of the reports in-

volve individuals for whom the traumatic event was exposure to combat, and 18 of these involve Vietnam veterans. Thus, over half of the published research on the psychophysiological assessment of PTSD involves Vietnam veterans who were, at the time they were studied, from about 11 to 25 years on average, post-trauma.

Given the findings of the National Vietnam Veterans Readjustment Study (Kulka et al., 1990), which estimated that, in the late 1980s, approximately 15 percent of those who served in southeast Asia still met the criteria for PTSD, or approximately 300,000 individuals, it is not surprising that this group has been a focal point of the research. Two other historical factors probably contributed to this focus: Kardiner (1941) in discussing the traumatic neuroses of war described a "physioneurosis" with most of the currently accepted symptoms of PTSD. Thus, those dealing with veterans had been sensitized to the issue for a long time. Second, the predecessor to the Department of Veterans Affairs, the Veterans Administration, officially (Gronvall, 1986) recognized the role of psychophysiological testing in diagnosing PTSD among veterans as early as 1986.

Two other traumatized groups have been the focus of limited psychophysiological research. There have been four reports on sexual assault survivors (SASs) and four reports on motor vehicle accident (MVA) survivors. Looking at epidemiologic studies focusing on PTSD (Norris, 1992; Kessler, Sonnega, Bromet, Hughes, and Nelson (1995), there are probably many more individuals with PTSD secondary to these two traumas than to combat.

#### WHAT HAS BEEN MEASURED?

Several different physiological responses have been measured, including heart rate (HR), blood pressure (BP), electrodermal activity (EDA) (either skin resistance or skin conductance), muscle activity (electromyogram [EMG]), peripheral temperature (surface temperature or sublingual temperature), and electroencephalogram (EEG). By far the most common response utilized is HR (29 of 31 studies), followed closely by EDA (18 studies) and EMG (16 studies), and finally BP (both systolic BP and diastolic BP).

 TABLE 12.1
 Studies of Psychophysiological Responding in War Time Trauma Populations

AUTHORS	POPULATIONS	YRS. POST TRAUMA	PROVOCATIVE STIMULI	RESPONSES
Dobbs & Wilson (1960)	Decompensated WWII vets (8) Compensated WWI, WWII, & Korean vets (13) Non-combat controls (10)	13 Years	Eight minute standardized audiotape of combat sounds accompanied by light flashes.	HR, EEG, RR
Blanchard et al. (1982)	Vietnam PTSD vets (11) Age matched non-vets (11)	11 Years	Five, 30-second, standardized audiotapes of combat sounds, alternated with 30-second trials of music. Decibel levels: 42, 52, 62, 72, 82.	HR, SBP, DBP, EMG, SC, PT
Malloy et al. (1983)	Vietnam PTSD vets (10) Vietnam Non-PTSD vets (10) Inpatient Non-PTSD Axis I (10)	14 Years	Sixty-second standardized videotaped presentations of 9 neutral scenes & 9 combat scenes with accompanying audio.	HR, SC
Blanchard et al. (1986)	Vietnam PTSD vets (57) Non-PTSD vets with similar exposure to combat (34)	15 Years	Five, 30-second standardized audiotapes of combat sounds, alternated with 30-second trials of music. Decibel levels: 40, 50, 60, 70, 80.	HR
Pallmeyer et al. (1986)	Vietnam PTSD vets (12) Non-PTSD Vietnam vets (10) Vietnam vets with Axis I (5) Era vets no disorder (5) Non-vets with anxiety disorder (8)	15 Years	Five, 30-second standardized audiotapes of combat sounds, alternated with 30-second trials of music. Decibel levels: 40, 50, 60, 70, 80.	HR, SBP, DBP, EMG, SC
Pitman et al. (1987)	Vietnam PTSD vets (18) Non-psychiatric Vietnam vets (15)	16 Years	Two, 30-second personal imagery audiotapes of traumatic war experience, with 1 standardized combat tape. Five personal imagery tapes of neutral, positive, action, & fear experiences.	HR, SC, EMG
Blanchard et al. (1989)	Vietnam PTSD vets (59) Non-PTSD vets with similar exposure to combat (12)	18 Years	Five, 30-second standardized audiotapes of combat sounds, alternated with 30-second trials of music. Decibel levels: 40, 50, 60, 70, 80.	HR
Gerardi et al. (1989)	PTSD Vietnam vets (18) Non-PTSD Vietnam vets (18)	18 Years	Five, 30-second standardized audiotapes of combat sounds, alternated with 30-second trials of music. Decibel levels: 40, 50, 60, 70, 80.	HR, SBP, DBP, PT, EMG, SC
Boudewyns & Hyer (1990)	Inpatient Vietnam vets PTSD (51)	19 Years	Three exposures to five-minute personal imagery audiotapes.	hr, emg, sc
Pitman & Orr (1990)	Vietnam PTSD vets (7) Vietnam non-PTSD vets with another anxiety disorder (7)	19 Years	Two, 30-second personal imagery audiotapes of traumatic war experience, with 1 standardized combat tape. Five personal imagery tapes of neutral, positive, action, & fear experiences.	hr, sc, emg
Blanchard, Kolb, Prins, et al. (1991)	Vietnam PTSD vets (15) Vietnam Non-PTSD vets (6)	20 Years	Three-minute standardized audiotape of combat sounds.	HR, Norep

AGE/ GENDER MATCHED	RESULTS	BASELINE DIFFERENCES	SENSITIVITY/ SPECIFICITY (METHOD OF CLASSIFICATION)
Age-No Gender-Yes	Decompensated vets. terminated procedures. Data available only for compensated vets and controls. Compensated vets > Controls on HR, RR, EEG.	Both groups of Vets > Controls on HR; Decomp. > Comp. > Controls on RR; Comp > Decomp. > Controls on EEG	None reported
Yes	PTSD > Controls on HR, SBP, EMG in response to provocative stimuli.	PTSD > Controls on HR (8 BPM)	91% vs. 100% (PDA using HR data)
Yes	PTSD > both control groups on HR reactivity to combat stimuli. Discriminant function analysis using 4 measures correctly classifies 80% of the sample.	None	80% vs. 80% (PDA)
Yes	PTSD > Controls on HR at every phase & greater reactivity to standardized combat stimuli.	PTSD > Controls on HR (12.6 BPM)	70% vs. 88% (Single cutoff score; the highest HR response to combat sounds)
No	PTSD > than all other control groups on baseline HR & responding to combat sounds.	PTSD vets higher than all other controls groups on HR	67% vs. 86% (PDA using HR responses)
Yes	PTSD > Controls on SC & EMG responses to personal traumatic imagery.	PTSD > Controls on resting HR (75 BPM vs. 66 BPM) $Eta^2 = .16$ ) <sup>a</sup>	100% vs. 61% (A discriminant function using all 3 responses)
Yes	PTSD > Controls on HR at every phase & greater reactivity to standardized combat stimuli.	PTSD > Controls on HR (10 BPM)	75% vs. 78% (Cutoff score of -1.0 for HR reactivity to combat stimuli minus HR reactivity to MA)
Age-No Gender-Yes	PTSD group was unable to suppress their physiological responses when asked to do so. Non-PTSD (n = 9) was able to fake physiological arousal.	None	70% vs. 88% (Using the single largest HR reactivity score)
N/A	Subjects showed increased arousal to audiotapes on all 3 physiological measures.	N/A	N/A
′es	PTSD > Controls on SC & EMG responses to personal traumatic imagery.	Anxiety do > PTSD on EMG ( $Eta^2 = .31$ )	71% vs. 100% (Using PDA derived by Pitman et al., 1987)
és .	PTSD > Controls on HR reactivity to combat stimuli. PTSD group showed 30% increase in Norepin upon exposure to traumatic cue.	None	Not reported

 TABLE 12.1 (Continued)

		YRS. POST		
AUTHORS	POPULATIONS	TRAUMA	PROVOCATIVE STIMULI	RESPONSES
Blanchard, Kolb & Prins (1991)	Vietnam PTSD vets (121) Derivation (69) Validation (52)  Vietnam Non-PTSD vets (79) Derivation (35) Validation (44)	20 Years	Five, 30-second standardized audiotapes of combat sounds, alternated with 30-second trials of music. Decibel levels: 40, 50, 60, 70, 80.	HR, SBP, DBP, EMG
McCaffrey et al.	Vietnam PTSD vets (5)	21 Years	Six standardized odors: orange, Peppermint,	EEG
(1993)	Vietnam Non-PTSD vets (5)	21 Icars	garlic, diesel fuel, burnt hair, decaying flesh.	220
McFall et al. (1992)	Vietnam PTSD vets (11) Vietnam non-PTSD vets (11)	21 Years	Baseline measurements only.	HR, SBP, DBP, Epin., Norepin
Orr et al. (1993)	WWI & Korean PTSD vets (8) Non-PTSD vets (12)	42 Years	Two, 30-second personal imagery audiotapes of traumatic war experience, with 1 standardized combat tape. Five personal imagery tapes of neutral, positive, action, & fear experiences.	HR, SC, EMG
Gerardi et al. (1994)	Vietnam PTSD vets (32) Era vets/no combat exposure (26)	23 Years	In Vivo assessment in an outpatient setting.	HR, SBP, DBP, RR, SL
Orr et al. (1995)	Vietnam PTSD vets (37) Vietnam Non-PTSD vets (19)	24 Years	Fifteen 95dB, 500-millisecond tones delivered via earphones.	HR, SC, (O)EMG
Davis et al. (1996)	Persian Gulf PTSD vets (14) Persian Gulf Non-PTSD vets (15)	3 Years	Two, thirty-second personal imagery scripts.	HR, EMG
Keane et al. (Under Review)	Vietnam veterans  Current PTSD vets (C) (778)  Lifetime PTSD (L) (181)  Never PTSD vets (N) (369)	25 Years	Twelve, standardized still images (6 combat & 6 neutral) with soundtrack of combat sounds. Two, thirty-second personal imagery audiotapes of trauma & 2 neutral tapes.	HR, SC, EMG, SBP, DBP
Muruoka et al. (Unpublished Dissertation)	Vietnam era PTSD vets (11) Vietnam era non-PTSD vets (7)	25 Years	Twenty-four hour ambulatory monitoring.	hr, SBP, DBP

AGE/ GENDER MATCHED	RESULTS	BASELINE DIFFERENCES	SENSITIVITY/ SPECIFICITY (METHOD OF CLASSIFICATION)
Yes	A predictive discriminant function derived from HR measures correctly identified 75% of the derivation sample & 80% of the validation sample.	Derivation PTSD HR 74.8 BPM NON HR 67.1 BPM	Derivation 84% vs. 57% Validation 85% vs. 82%
		Validation PTSD HR 76.9 BPM NON HR 67.7 BPM	(PDA using HR measures)
Yes	PTSD showed a marked increase in left hemisphere activity relative to Controls upon exposure to burnt hair stimuli.	Not reported	Not reported
Yes	No statistically significant differences on any measure across the 4 baseline measurements.	None	Not reported
Yes	PTSD > Controls on HR & SC responses to personal traumatic imagery.	None	88% vs. 100% (Using discriminant function derived by Pitman, Orr, Forgue, et al., 1990)
Yes	PTSD > Controls on resting HR, SBP, & DBP.	PTSD > Controls on resting HR, SBP, & DBP	Not reported
Yes	PTSD vets showed greater (O)EMG & HR responses to stimuli & less diminution of SC across phases.	None	Not reported
Age-Yes Gender-Yes	Statistical trends for the PTSD group to be more responsive than controls on both EMG & HR.	PTSD HR = $73.2$ BPM Control HR = $67.7$ BPM Eta <sup>2</sup> = .11	Not reported
Age-No Gender-Yes	Audiovisual C > L, N on HR response C > L, N on SC C > N on DBP Imagery Scripts C, L > N on HR C > N on SC C, L > N on EMG Logistic Regression using psychophysical measures correctly classifies 69% in the derivation sample & 64% in the validation sample.	C > L, N on HR C > L on SC	Derivation 83% vs. 42% Validation 81% vs. 31% (Logistic Regression)
Age-No Gender-Yes	PTSD > non-PTSD vets on overall HR & DBP measures.	Differences were seen during waking & sleeping hours	Not reported

Note. PTSD = Posttraumatic Stress Disorder; HR = Heart Rate; SBP = Systolic Blood Pressure; DBP = Diastolic Blood Pressure; SC = Skin Conductance; EMG = Frontalis Electromyogram; (O)EMG = Orbicularis Electromyogram; PT = Peripheral Temperature; RR = Respiration Rate; Norepin. = Norepinephrine; SL = Sublingual Temperature; Epin = Epinephrine; PDA = Predictive Discriminant Analysis; MVA = Motor Vehicle Accident; BPM = Beats Per Minute; MA = Mental Arithmetic

<sup>a</sup>Eta<sup>2</sup> is an effect size measure that is a proportion of variance accounted for. Thus, multiplying by 100 would give the percentage of variance accounted for by independent variables.

 TABLE 12.2
 Studies of Psychophysiological Responding in Civilian Trauma Populations

AUTHORS	POPULATIONS (N)	YRS. POST TRAUMA	STIMULI	RESPONSES
Blanchard, Hickling et al. (1991)	MVA related PTSD (4)	7.5 Months	Two, 3-minute personal imagery audiotapes of MVA.	HR, SBP, DBP, SC
Blanchard et al. (1994)	MVA victims (50) PTSD (23) Sub-PTSD (10) Non-PTSD (17) Non-MVA controls (40)	2.5 Months	Two, 3-minute personal imagery audiotapes of MVA. One, standardized, 2-minute video of MVAs.	HR, SBP, DBP, EMG, SC
Bryant et al. (1995)	MBA related PTSD (10) Non-MVA normals (10)	3.25 Years	Stroop Threat word.	SC
Blanchard et al. (1996)	MVA victims (105) PTSD (38) Sub-PTSD (35) Non-PTSD (32) Non-MVA controls (54)	2.5 Months	Two, 3-minute personal imagery audiotaped descriptions of MVA. One, standardized, 2-minute video of MVAs.	hr, sbp, dbp, emg
Kilpatrick et al. (1984)	Txseeking rape victims (27)	1.25 Years	Five 1-minute personal imagery audiotapes: 1-neutral scene, 1-positive scene, 3 fear-evoking rape-related scenes.	HR, SC
Kozak et al. (1988)	PTSD rape victims (12) No rape controls (12)	Range 6 Weeks–2 Years	Standard neutral scene Standard rape scene in 30-second audiotaped format	HR, SC
Griffin et al. (1994)	Recent rape victims (90) (Sample broken down into High- Medium-Low dissociators which served as Independent Variables)	2 Weeks	Five-minute netural discussion with therapist and 5-minute trauma discussion.	HR, SC
Forneris et al. (1996)	Rape victims (13) PTSD (3) Sub-PTSD (3) Non-PTSD (7) Non-rape controls (13)	Not Reported	Two-minute personal imagery audiotapes.	HR, EMG, SBP, DPT, SC
Shalev, Orr, Peri, et al. (1992)	Civilian trauma PTSD (14) Anxiety DO (14) Normals previous trauma (15) Normals w/o previous trauma (19)	5.75 Years	Fifteen 95dB, 500-millisecond tones delivered via earphones.	HR, SC, (O)EMG
Shalev et al. (1993)	Israeli civilian trauma PTSD (13) Non-PTSD trauma civilians (13)	4.6 Years	Two, 30-second personal imagery audiotapes of traumatic war experience, with 1 standardized combat tape. Five personal imagery tapes of neutral, positive, action, & fear experiences.	HR, SC, EMG

AGE/ GENDER MATCHED	RESULTS	BASELINE DIFFERENCES	SENSITIVITY/SPECIFICITY (METHOD OF CLASSIFICATION)
N/A	Increases over baseline levels on HR for 3 of 4 (9.2 BPM) also for SBP (4 of 4) & 2 of 4 for EDA.	All were within the normal HR range at baseline except 1.	Not applicable
Yes	MVA-PTSD group was more responsive on HR measure upon presentation of idiosyncratic audiotapes relative to non-PTSD MVA victims & controls.	None	74% vs. 76% (Single cutoff score of 2 BPM when responding to personal imagery)
Yes	PTSD > Controls on responses to neutral & threat words.	None	Not reported
Yes	MVA-PTSD group was more responsive on HR measure upon presentation of idiosyncratic audiotapes relative to non-PTSD	None	69% vs. 78%  (Single Cutoff score of 2 RPM)
	MVA victims & controls. Strong initial HR response predicted poor clinical outcome 1 year later for PTSDs.		(Single Cutoff score of 2 BPM increase to Personal imagery)
N/A	HR and SC responding was greater relative to baseline for most conditions. Pleasant scene was just as effective as fear-evoking scenes in eliciting a response.	As a group, baseline hyperarousal was not evident.	Not reported
Yes	Trends for greater responding of PTSD group on both HR & SC measures. Failed to reach statistical significance.	Not reported	Not reported
N/A	High dissociators respond with lower SC responses that low or medium dissociators.  No group effects on HR data.	Not Reported	Not reported
Yes	No significant differences between PTSD and non-PTSD groups.	None	Not reported
Age-Yes Gender-No	PTSD group showed larger HR & SC responses relative to the other groups. They were the only group that failed to habituate across trials.	None	Not reported
No	PTSD > Controls on HR & EMG responses to personal traumatic imagery.	Non > PTSD on SC	69% / 77% (Using PDA derived by Pitman et al. 1987)

Note. PTSD = Posttraumatic Stress Disorder; HR = Hear Rate; SBP = Systolic Blood Pressure; DBP = Diastolic Blood Pressure; SC = Skin Conductance; EMG = Frontalis Electromyogram; (O)EMG = Orbicularis Electromyogram; PT = Peripheral Temperature; Norepin. = Norepinephrine; SL = Sublingual Temperature; Epin = Epinephrine; PDA = Predictive Discriminant Analysis; MVA = Motor Vehicle Accident; BPM = Beats Per Minute

The two most commonly used responses, HR and EDA, are good indicators of sympathetic nervous system involvement (although HR is controlled jointly by sympathetic and parasympathetic input). Exploring the sympathetic nervous system underpinnings a bit further, Blanchard, Kolb, and Prins (1991) found increases in plasma norepinephrine (accompanied by elevated HR) among Vietnam veterans with PTSD compared to similar combat veterans without PTSD, following exposure to an audiotape with combat sounds. This tonic change in the biochemical substrate was consistent with Mason, Giller, Kosten, Ostroff, and Podd (1986) findings of higher 24-hour urinary levels of norepinephrine and cortisol in Vietnam veterans with PTSD. Pitman and Orr (1990), however, failed to replicate Mason et al.'s findings. One would expect to see BP responses follow a similar pattern to those found for HR.

The EMG responses were primarily from a frontal (forehead) or frontalis placement but two studies involved EMG measures of obicularis oris associated with orienting and startle responses. These skeletal muscle responses show that arousal goes beyond the autonomic nervous system.

#### WHAT IS THE BEST RESPONSE TO USE?

This question addresses both the research summary issue and a very practical issue. Taking a box score approach, when HR and EDA have both been used in the same study (n = 17), HR has apparently yielded significant results in 12 instances when EDA did not, while EDA has yielded significant findings on three occasions when HR was not significant. On two occasions both yielded significant results. (One can also address this question more precisely with the results in Table 12.3, a summarization of the values for variance accounted for by the different measures in different studies. See below.)

Overall, of the 29 studies which have utilized HR, 21 (72.4 percent) found significant results. For the 18 studies using EDA, 8 (44.4 percent) found significant results. Finally, for the 16 studies using EMG, 8 (50 percent) found significant results.

#### **Clinical Hint**

Our own experience has been that EMG, especially frontal EMG, is not very useful. We recommend dropping it from psychophysiological assessments unless one has an especially compelling reason to include it. Again, from our experience, we would choose HR over EDA if one is forced to use a single physiological channel. Our preference would be to use both HR and EDA.

Temperature is a very slow response and does not appear useful. Blood pressure is certainly a very important health parameter and should be added, if possible, especially with a middle-aged or older male population.

## WHAT FORMS OF STIMULUS PRESENTATION HAVE BEEN USED AND WHICH ARE PREFERABLE?

The overwhelming favorite for stimulus modality is auditory with 24 reports having an audio stimulus of one fashion or another, while three used solely visual stimuli, and three an audiovisual combination. One reason so many investigators may have chosen auditory stimuli is that it is difficult for the research participant to voluntarily shut off the stimulus presentation when it is auditory (one can close one's eyes to a visual stimulus but one cannot close one's ears).

Two reports (Blanchard et al., 1996; Keane et al., 1996) have compared auditory and visual traumatic stimulus presentations. One intriguing report (McCaffrey, Lorig, Pendrey, McCutcheon, & Garrett, 1993) used olfactory stimuli. We suspect using cues in the chemical senses (olfactory or gustatory) may be very powerful because these sensory channels are very old phylogenetically and make almost direct connections with the midbrain. Anecdotally, we continue to hear from patients how powerful chemical sense reminders can be for those with PTSD. More research is clearly needed on this topic.

Interestingly, very little work has been done comparing one stimulus modality to another. The three comparisons (Keane et al., 1996; Blanchard, Hickling, Taylor, Loos, & Gerardi, 1994; Blanchard

 TABLE 12.3
 Effect Sizes (Eta²)a of Psychophysiological Responding in Trauma Populations

AUTHORS	POPULATIONS (N)	ETA <sup>2</sup> REACTIVITY SCORES TO PROVOCATIVE STIMULI	ETA <sup>2</sup> BETWEEN-GROUPS COLLAPSED ACROSS ALL EXPERIMENTAL PHASES
Blanchard et al. (1982)	Vietnam PTSD Vets (11) Age matched non-Vets (11)	Not reported	HR = .16 SBP = .23 DBP = .15 SC = .15 EMG = .00 PT = .00
Blanchard et al. (1986)	Vietnam PTSD vets (57) Non-PTSD vets with similar exposure to combat (34)	HR = .25 (Heart rate response to 60db combat sounds)	
Blanchard et al. (1989)	Vietnam PTSD vets (59) Non-PTSD vest with similar exposure to combat (12)		$HR = .63^{b}$
Blanchard et al. (1994)	MVA victims (50) PTSD (23) Sub-PTSD (10) Non-PTSD (17) Non-MVA controls (40)	HR = .19	
Blanchard et al. (1996)	MVA victims (105) PTSD (38) Sub-PTSD (35) Non-PTSD (32) Non-MVA controls (54)	HR = .11 SBP = .02	
Davis et al. (1996)	Persian Gulf PTSD vets (14) Persian Gulf Non-PTSD vets (15)	HR = .11 EMG = .02	
Gerardi et al. (1989)	PTSD Vietnam vets (18) Non-PTSD Vietnam vets (18)		HR = .16 DBP = .29
Gerardi et al. (1994)	Vietnam PTSD vets (32) Era vets/no combat exposure (26)	HR = .13 SBP = .08 DBP = .12 SL = .01 RR = .04 (Baseline Only)	
Griffin et al. (1994)	Recent rape victims (90) (Sample broken down into High-Medium-Low dissociators which served as Independent Variables)	SC = .08	
Keane et al. Under Review)	Vietnam veterans Current PTSD vets (C) (778) Lifetime PTSD (L) (181) Never PTSD vets (N) (369)	Audio HR = .03 SC = .03 SBP = .01 DBP = .01 EMG = .01	

(Continued)

TABLE 12.3 Continued

AUTHORS	POPULATIONS (N)	ETA <sup>2</sup> REACTIVITY SCORES TO PROVOCATIVE STIMULI	ETA <sup>2</sup> BETWEEN-GROUPS COLLAPSED ACROSS ALL EXPERIMENTAL PHASES
		Video HR = .02 SC = .02 SBP = .01 DBP = .01 EMG = .02	
Muroaka et al. (Unpublished Dissertation)	Vietnam era PTSD vets (11) Vietnam era Non-PTSD vets (7)	HR = .20 DBP = .28 SBP = .05 (24-hour amubulatory measures only)	
Orr et al. (1993)	WWII & Korean PTSD vets (8) Non-PTSD vets (12)	HR = .45 SC = .40 EMG = .25	
Orr et al. (1995)	Vietnam PTSD vets (37) Vietnam Non-PTSD vets (19)		(O)EMG = .10 SC = .01 HR = .14
Pallmeyer et al. (1986)	Vietnam PTSD vets (12) Non-PTSD Vietnam vets (10) Vietnam vets with Axis I (5) Era vets no disorder (5) Non vets with anxiety disorder (8)		HR = .24 SBP = .28 DBP = .03 SC = .18 EMG = .20
Pitman et al. (1987)	Vietnam PTSD vets (18) Non-psychiatric Vietnam vets (15)	SC = .27 EMG = .14 HR = .08	
Pitman et al. (1990)	Vietnam PTSD vets (7) Vietnam Non-PTSD vets with another anxiety disorder (7)	EMG = .32 SC = .27 HR = .12	
Shalev et al. (1992)	Civilian trauma PTSD (14) Anxiety DO (14) Normals previous trauma (15) Normals w/o previous trauma (19)		(O)EMG = .03 HR = .28 SC = .44
Shalev, Orr, Peri, et al. (1993)	Israeli Civilian trauma PTSD (13) Non-PTSD trauma civilians (13)	HR = .33 EMG = .25 SC = .08	

Note. PTSD = Posttraumatic Stress Disorder; HR = Heart Rate; SBP = Systolic Blood Pressure; DBP = Diastolic Blood Pressure; SC = Skin Conductance; EMG = Frontalis Electromyogram; (O)EMG = Orbicularis Electromyogram; RR = Respiration Rate, MVA = Motor Vehicle Accident

<sup>&</sup>lt;sup>a</sup>Eta<sup>2</sup> is an effect size measure that is a proportion of variance accounted for. Thus, multiplying by 100 would give the percentage of variance accounted for by independent variables.

<sup>&</sup>lt;sup>b</sup>The data used to calculate the effect size measure for Blanchard et al., 1989, is based on statistics that utilized the combined sample of Blanchard et al., 1986 & 1989.

et al., 1996) all confounded stimulus modality with the standard stimulus versus idiosyncratic stimulus difference.

There is another dimension to stimulus presentation which has been addressed in this research: whether to use a standardized (or generic) stimulus, that is, the same stimulus is presented to all individuals in the research (with the assumption that the generic stimulus captures some part of each participant's experience of the trauma) or to use an idiosyncratically tailored stimulus for each participant (with the stipulation of the rules for creating these individualized stimuli). The former approach was used by Blanchard, Kolb, and their associates in their studies with Vietnam veterans with PTSD. An audiotape (taken from the sound track of Apocalypse Now) was played at progressively louder sound intensities (roughly 40, 50, 60, 70, and 80 decibels) for 30second intervals. Sounds included AK-47 fire, mortars, helicopters, and screams of the wounded.

A variation of this was used by Malloy, Fairbank, and Keane (1983) who combined still pictures showing progressively more hazardous combat engagement with a sound track of accompanying combat sounds played at progressively louder volumes. Again, the same stimuli were used for all participants. Blanchard and colleagues (Blanchard et al., 1994; Blanchard et al., 1996) used a standardized videotape of car crashes, including scenes shot from inside the vehicle as a crash was occurring, in their work with motor vehicle accident survivors. Finally, Kozak, Foa, Olasov-Rothbaum, and Murdock (1988) used standardized 30-second audiotape descriptions of rape in their work.

Other stimulus modalities (such as Stroop words related to MVAs [Bryant, Harvey, Gordon, & Barry, 1995]) have had only a single paper reporting their usage.

The idiosyncratic approach was pioneered by Pitman and Orr (Pitman, Orr, Forgue, de Jong, & Claiborn, 1987), who used 30-second audiotaped descriptions of idiosyncratic trauma experiences. They acknowledged an intellectual debt to Peter Lang and his work on stimulus propositions and response propositions in fear arousing imagery (Lang, 1979). The

versatility of this approach was shown in Israeli studies by Shalev, Orr, and Pitman (1993) in a study of victims of varying traumatic experiences (motor vehicle accidents, sexual assaults, physical assaults, terrorist attacks, and witnessing violence). Pitman and Orr have also included a standard audio description in several of their studies.

A box score approach reveals 14 reports have used standardized stimulus presentations, almost all of which were with Vietnam combat veterans, versus 14 reports which have used idiosyncratic presentations. Three reports (Blanchard et al., 1994; Blanchard et al., 1996; Keane et al., 1996) have used both idiosyncratic presentations and standardized stimuli with MVA survivors, MVA survivors, and Vietnam veterans, respectively. Both of Blanchard's MVA studies found idiosyncratic audiotapes more arousing (and thus better able to discriminate participants with PTSD from comparison groups) than standardized videotapes of car crashes. Keane et al. (1996) found the standardized audio-visual stimulus and the idiosyncratic audiotapes equally provocative with Vietnam combat veterans.

Although one of us (EBB) clearly showed a preference for the standardized stimulus early on, he has come to favor the idiosyncratic audiotape stimulus because of its flexibility and its ability to be readily adapted to subtle personal nuances of trauma experience in the individual case. As Tables 12.1 and 12.2 point out, there are reports of successful use of idiosyncratic audio stimuli with various combat veteran groups from World War II, Korean War, and Vietnam War, up to Operation Desert Storm, as well as sexual assault and rape victims, other assault victims, and motor vehicle accidents. In all of these groups of victims of diverse trauma, the idiosyncratic audiotape has proved itself.

Length of description has varied from the very precise 30-second tapes of Pitman and Orr to 2 to 3 minute descriptions used by Blanchard and colleagues with MVA survivors and by Forneris (1996) with sexual assault victims to 5 minute discussions of trauma with rape victims used by Griffin, Resick, and Mechanic, (1994) and 5-minute descriptions of combat scenes by Boudewyns and Hyer (1990).

### BASELINE DIFFERENCES AND AMBULATORY PSYCHOPHYSIOLOGICAL MEASUREMENT

One can note that both Table 12.1 and Table 12.2 have columns for comments devoted to possible baseline differences in psychophysiological measures between participants with PTSD and the comparison groups. One of us (EBB) (Blanchard, 1990) called attention several years ago to the apparent baseline differences in cardiovascular responses (HR, SBP, and DBP) among Vietnam veteran groups of comparable age who either met criteria for PTSD or did not. In that report, the average difference in resting HR was 10.3 bpm while the average differences in both SBP and DBP were about 7 mm of mercury.

Subsequent reports on combat veterans, including Keane et al.'s (1996) large study of Vietnam veterans and Davis, Adams, Uddo, Vasterling, and Sutker's (1996) report on Operation Desert Storm veterans, have continued to report significant baseline differences on HR of about the same magnitude. There have also been a few reports of differences in resting EDA (Keane et al., 1996; Malloy et al., 1983).

In other traumatized populations (Table 12.2), the general rule has been an absence of baseline differences between trauma survivors with PTSD and comparison groups. Shalev et al. (1993), in the assessment of victims of various traumas, found significant resting baseline differences in skin conductance with the stress exposed PTSD negative group having a higher skin conductance than the PTSD group. This would seem to indicate more basal sympathetic arousal in the non-PTSDs.

Blanchard (1990) speculated on two possible explanations for the basal cardiovascular differences among combat veterans: (1) It could be the case that those with PTSD could be in a relatively permanent state of "sympathetic overdrive" and thus the baseline difference represents a true difference. (2) Alternatively, it could be that those with PTSD, having some foreknowledge of the nature of the assessment, i.e., exposure to combat stimuli (due to the necessity for informed consent), were aroused in anticipation of the psychophysiological assess-

ment and the adaptation and baseline conditions were not sufficiently long to allow this arousal to dissipate. In other words, the difference was an "experimental artifact." 1

The first attempt to examine this issue was the study by McFall, Veith, and Murburg (1992), which took extended baseline (no provocative combatrelated stimulation) measures of HR, SBP, DBP, plasma norepinephrine, and epinephrine on 11 Vietnam veterans with PTSD and 11 matched veterans without PTSD. They found no significant differences on any measure, lending some credence to the "experimental artifact" explanation.

Gerardi, Keane, Cahoon, and Klauminzer (1994) examined the medical records of 32 Vietnam veterans with PTSD and 26 Vietnam era veterans who never served in Southeast Asia. Both groups were seeking services, medical or psychological, at a VA hospital. HR, BP, and RR were taken by the triage nurse as part of admission, and recorded in the patients' charts. Comparisons revealed significantly higher resting HR (89 versus 78 bpm), SBP (133 versus 124 mm Hg), and DBP (88 versus 79 mm Hg) but no difference in respiration rate. These veterans were not assessed as part of a formal psychophysiological assessment; thus the experimental expectation was absent. Despite this situation, the basal differences were present.

Finally, in a very elegant study, Muruoka, Carlson, and Chemtob (1995) assessed 11 Vietnam veterans with PTSD and 7 comparable veterans without PTSD using 24-hour ambulatory monitoring. Across the 24 hours, the veterans with PTSD had higher average HR (81 versus 72 bpm) and DBP (80 versus 72 mm mercury). Moreover, HR during sleep was significantly greater for veterans with PTSD (71 bpm versus 63 bpm). These results seem to confirm that, at least among veterans with PTSD, there is a significant elevation in cardiovascular responses and that these patients are more aroused all of the time than veterans without PTSD.

<sup>&</sup>lt;sup>1</sup>The NIMH in its wisdom never supported Blanchard and Kolb efforts to explore this issue. Fortunately, others have done so.

### IS ONE PHYSIOLOGICAL RESPONSE SYSTEM MORE SENSITIVE THAN OTHERS?

Earlier, we summarized the preferences of various research groups for one physiological response over another in terms of frequency of use, a relatively qualitative analysis, and counted instances of significant results. One can see from Tables 12.1 and 12.2. that physiological responding to cues reminiscent of trauma is evident across a multitude of survivor populations. Moreover, the number of independent sites that have replicated the effects is quite impressive. To further understand how well these measures distinguish PTSD populations from non-PTSD populations, we have calculated effect sizes (eta squared, eta<sup>2</sup>) for all published reports that provided adequate statistical information to do so and summarized them in Table 12.3. Eta<sup>2</sup> is an effect size measure that is a proportion of variance accounted for by independent variables. Thus, multiplying the values in the table by 100 would give one the percentage of variance accounted for by those independent variables (which, in this case, is diagnostic group membership).

For the sake of brevity, we have not arranged the studies into separate tables by trauma type. Rather, we organized the experimental studies (previously described in Tables 12.1 and 12.2) in Table 12.3 alphabetically by first author, and chronologically within author.

The effect size that is of greatest interest is the between-group effect size of physiological responding to provocative stimuli that are reminiscent of one's trauma. This effect size most adequately captures the essence of symptom number five, "physiological reactivity on exposure to internal or external cues that symbolize or resemble an aspect of the traumatic event," of the DSM-IV (APA, 1994) diagnostic criteria for PTSD. This effect size appears in column three. However, in some reports, this effect size could not be computed. In some of these cases, statistics were only available to compute eta<sup>2</sup> on the group mean differences across all phases of the psychophysiological assessment (i.e., baseline, neutral stimuli phases, and provocative stimuli). Thus, in these cases, eta<sup>2</sup> represents the proportion of variance accounted

for by the independent variables of group membership (PTSD versus controls) across all phases, not just the reactivity scores to provocative stimuli. These values are presented in column four.

We mentioned previously in this paper that several published reports have opted for standardized stimulus presentations, while others have utilized an idiosyncratic or personalized imagery approach. Of particular interest to us, was whether or not effect sizes differed as a function of stimulus presentation. For the six studies that utilized standardized auditory stimuli (one study used loud tones), the mean eta<sup>2</sup> value for HR was .33 as opposed to .17 for those studies employing personalized imagery scripts. For EMG, the standardized auditory stimuli approach yielded a mean eta<sup>2</sup> value of .10, while the personal imagery approach yielded a mean value of .16. For studies that measured EDA, the standardized approach yielded a mean eta<sup>2</sup> value of .26, while the personal imagery approach yielded a value of .16 (unfortunately, there were not enough studies across the two types of presentations to make comparisons on the other physiological channels). Based on these values, one might tenuously conclude that the type of stimulus presentation affects physiological channels in different ways. However, this statement should be approached with caution given the nature of the calculations. First, not all studies reported adequate information for the computation of eta<sup>2</sup>, thereby reducing the accuracy of the conclusions drawn from these data. Moreover, some of the eta<sup>2</sup> values used were not on reactivity scores to provocative stimuli, but on group effects across all experimental phases. Finally, these data were collapsed across trauma type. Different conclusions may be drawn if effect sizes are compared within trauma populations. Finally, it could be the case that time since trauma (and thus, time with PTSD) may be linked to effect sizes. As suggested by Kolb (1987), those with chronic PTSD of many years may actually develop disordered sympathetic nervous systems. If this is the case, one would expect acute populations to respond differently than chronic populations. Thus, we suggest that the personal versus standardized approach is an interesting area for future research. Based on personal experience we would

endorse the use of idiosyncratic audiotapes as the preferred stimulus presentation method.

Another interesting question one could ask is whether or not the modality of stimulus presentation matters. For example, are the effect sizes larger or smaller for video presentations of stimuli or audio presentations? Unfortunately, there are only three studies that have used both methods; however, all three confounded stimulus modality with personal versus standardized imagery. Of course, the use of visual presentation of stimuli is somewhat limiting in the sense that all stimuli have to be standardized by definition. It would be very difficult to create "personalized" video stimuli reminiscent of an individual's trauma. Again, we suggest this might be an interesting question to be answered by future research that employs standardized stimuli.

One study in Table 12.3 stands out for the relatively low values of eta<sup>2</sup>, that of Keane et al., 1996.

### THE VA COOPERATIVE STUDY (KEANE ET AL., 1996)

By far the most ambitious study of the role of psychophysiological assessment in PTSD is that of Keane et al. (1996). This paper reported on VA Cooperative Studies Project No. 334 by Kolb and Keane (1988) (Psychophysiology Study of Chronic Post-Traumatic Disorder). In this project detailed diagnostic data and psychophysiologic assessment data were gathered on 1,328 veterans who served in Southeast Asia during the Vietnam War era 1963-1973. They were carefully characterized, based upon multiple measures, into 3 groups: 778 who currently met criteria for PTSD, 181 who had been positive for PTSD at some point in their past lifetime, but who currently were not positive for PTSD, and 369 who never met the criteria for PTSD. Five psychophysiological responses were measured. Both the Pitman-Orr idiosyncratic audiotapes were used as was the audio-visual combination stimulus of Malloy et al. (1983).

Results showed significant discrimination between current PTSDs and the other two groups with the standardized audio-visual stimulus and HR and skin conductance. There was significant discrimination between the current PTSD and never PTSD with the standardized audio-visual stimulus and DBP and with idiosyncratic audiotapes and skin resistance. The level of sensitivity and specificity were significant but relatively low.

An important part of this paper was that the non-PTSDs and current PTSDs were selected from the same population, treatment-seeking veterans from the VA. As such, they were more similar on many dimensions than comparable groups in other studies. In almost all other studies, the non-PTSD combat veterans were selected from volunteer, usually non-VA, populations.

Unfortunately, the degree of identification in this study at the level of the individual subject is such that the clinical utility of psychophysiological measures is somewhat called into question. At a practical level, this paper seems to show that HR and skin conductance responses to a standardized audio-visual presentation may be best for assessing Vietnam veterans.

### FOR WHAT IS PSYCHOPHYSIOLOGICAL ASSESSMENT USEFUL?

At this point, psychophysiological assessment's main role in the assessment of PTSD seem to be one of confirmatory adjunct. All of the research to date relies upon some form of interview, structured or otherwise, as the "gold standard" to determine definitively whether a subject has PTSD. Most of the research has been designed (1) to see whether PTSDs are more responsive than those without current PTSD at the level of group mean differences (the answer is usually "yes") or (2) to see how well some single variable or set of variables can discriminate those with PTSD from comparison groups. Separation has ranged from 100 percent correctly classified, Malloy et al. (1983), to 65 percent correctly classified. At the high end, psychophysiological testing could begin to substitute for the structured clinical interview. At the low end, the accuracy is such that one would be uncomfortable making clinical decisions on its basis alone. Even with relatively poor discrimination, however, the psychophysiological assessment data serves as a useful adjunct in the overall assessment of PTSD, a point made over ten years ago by the VA (Gronvall, 1986).

#### "Truth Detection" and Dissimulation

A hope held by many in this field was that psychophysiological testing might serve as a non-verbal "truth detector." It is fairly easy for a motivated individual to learn the symptomatic criteria for PTSD. If such an individual has the appropriate history of exposure to trauma, it is readily possible for that individual to feign PTSD. Psychophysiological testing might help detect the dissemblers, but probably not with the accuracy one would hope to take to court or a disability rating board.

Gerardi, Blanchard, and Kolb (1989) assessed 18 Vietnam combat veterans with PTSD and 18 comparable Vietnam combat veterans without PTSD using the standardized audiotape of combat sounds (30-second exposures at progressively higher sound levels) while measuring HR, SBP, DBP, forehead EMG, and EDA (as skin resistance level). The initial assessment yielded the usual results with good discrimination between PTSD and non-PTSD groups. Again, HR was the single best response, correctly identifying 80.6 percent of the sample (p = .0001).

Next, half of the randomly selected veterans without PTSD were given a description of the typical pattern of responses of those with PTSD and were asked, in a second assessment a few minutes later, to try to simulate the responding of someone with PTSD. They were given explicit information on how responses should change (e.g., increased HR). Comparisons of the simulators to those with PTSD, also assessed a second time, revealed that only DBP reactivity scores discriminated between the two groups (p < .01). Discriminant functions were still significant for HR, DBP, SBP, and frontal EMG, but not EDA. The best overall discrimination of veterans with PTSD from veterans without PTSD who were attempting to fake the response comes from a combination of baseline HR and maximum HR response to combat sounds. This correctly identified 89 percent of veterans with PTSD and 67 percent of those attempting to simulate PTSD.

Orr and Pitman (1993) assessed 25 Vietnam combat veterans with PTSD and 18 comparable veterans without PTSD using their 30-second idiosyncratic audiotape stimuli and HR, EDA (as skin conductance), and three different facial EMG measures. Seven weeks later the non-PTSDs were reas-

sessed and asked to produce responses like someone with PTSD by "getting yourself emotionally 'worked up.'" Thus, the instructions were less explicit and the interval considerably longer.

At the initial session a discriminant function using skin conductance and corrugator EMG correctly identified 18 of 25 PTSDs and 16 of 16 non-PTSDs. When this discriminant function was applied to the second (simulation) session data of those without PTSD, 4 subjects were identified as PTSD positives and 12 were identified as non-PTSD. In effect, 75 percent were correctly identified.

Clearly, more research is needed on this important topic. There appear to be possible instruction and practice effects which one would want to tease out.

#### **Prediction of Future Clinical Status**

An obvious question to address with psychophysiological assessment results is whether they predict future clinical status, with or without intervening treatment. One study has addressed this issue explicitly: Blanchard et al. (1996) identified 48 motor vehicle accident survivors who met the criteria for PTSD 1 to 4 months post-accident. At an assessment 12 months later 16 (33 percent) had not remitted whereas 32 had remitted totally or in part. The HR response of these 48 individuals to idiosyncratic audiotapes of their accidents had been recorded at the initial assessment. These data correctly identified 37 (of 48) individuals as still having PTSD (11/16) or as remitting (26/32) 12 months later. Other work on the prediction of remission or response to treatment is clearly needed. However, we are impressed by this level of prediction from a single variable.

## Within-Treatment Session Psychophysiological Data

In addition to pre-treatment physiological measures being used to predict long-term clinical outcome, physiological measures taken during treatment phases may be useful in assessing the adequacy of behavioral strategies used to target specific symptoms of PTSD. It has been suggested by Foa and Kozak (1986) that in order for exposure based therapies of anxiety disorders to work, therapist's need to tailor

exposure exercises such that they are effectively tapping into the patients "fear network." Theoretically, when an individual's fear network is tapped, there should be a strong anxiety response that can be directly measured through physiological responses. If one fails to elicit this response, exposure will presumably not be successful. Thus, Foa and Kozak suggest that exposure exercises should be set up to achieve maximum physiological arousal in order for them to be successful. In general, one should see within-session decreases in physiological responses (habituation) during treatment if exposure is working effectively. In addition, one should also see betweensession decrements in physiological responding if the exposure based therapy is working effectively. Thus, the theory proposed by Foa and Kozak is elegant in the way that it readily lends itself to being tested through the use of the physiological measures that have been shown in this chapter to readily distinguish PTSD positives and negatives (for a detailed account of propositional network theory and processing of fearful stimuli, see Foa & Kozak, 1986).

In fact, four treatment studies of PTSD addressed the issue of whether physiological responding is linked to treatment outcome. Shalev, Orr, and Pitman (1992), treated 3 cases of civilian related PTSD. Elevated physiological responses to traumatic imagery prior to treatment were treated with systematic desensitization. Physiological responding to these images was diminished at post-treatment. All patients showed improvement in overall PTSD symptoms. However, traumatic stimuli that were not used as exposure based exercises during treatment still elicited elevated physiological arousal at post-treatment.

Fairbank and Keane (1982) successfully treated a case of combat related PTSD with an exposure based therapy. Heart rate and skin conductance data taken at each session showed that responding decreased within each session after exposure. In addition, between-session decrements in overall responding were also apparent. One other case study of successful

treatment of combat related PTSD (Keane & Kaloupek, 1982) provided data that showed pre- to post-treatment decrements in physiological responding (within-session data was not obtained). Boudewyns and Hyer (1990) treated 51 cases of combat related PTSD with either an exposure based therapy or conventional one-on-one counseling. As a whole, both groups showed significant physiological responding at both pre- and post-treatment. However, at an individual level, subjects who showed decreased physiological arousal at post-treatment improved at three-month follow-up when compared to those subjects who did not show decreased responding at follow-up.

These case studies and the results provided by Boudewyns and Hyer (1990) provide data that are consistent with the theory put forth by Foa and Kozak (1986). Given the current state of affairs in the health care system, with a focus of designing effective treatments that are readily translated into good outcome measures, psychophysiological correlates of treatment outcome appear to present a fruitful area of PTSD treatment outcome research.

#### **CONCLUSIONS**

Psychophysiological assessment, especially heart rate and to a lesser degree, electrodermal activity responses, measured to idiosyncratic audiotapes reminding the participant of his or her traumatic experience, appears to be useful with combat veterans and motor vehicle accident survivors, and to those with mixed trauma. It is apparently less useful in the assessment of sexual assault survivors. In the main, psychophysiological assessment can serve as a useful adjunct to standardized clinical interviews by helping to confirm PTSD diagnoses. Psychophysiological assessment may also have a very valuable role to play both in the prediction of long-term outcome to stress exposure and also in evaluating short-term outcome of treatments for PTSD.

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